Proceedings of the 60th Annual Convention of the American Association of Equine Practitioners - AAEP –

December 6-10, 2014
Salt Lake City, UT, USA

Next Meeting:
Dec. 3-7, 2016 - Orlando, FL, USA

Reprinted in the IVIS website with the permission of the AAEP
http://www.ivis.org
Auditory Loss in Horses, Part I: Adult Horses

Monica Aleman, MVZ Cert., PhD, DACVIM (Internal Medicine, Neurology)*; Terrell A. Holliday, DVM, PhD, DACVIM (Neurology); Jorge E. Nieto, MVZ, PhD, DACVS, DACVSMR; and Colette Williams, PhD

Evaluation of hearing should be part of the physical and neurological examination since hearing loss occurs in horses and could result in altered behavior and performance. If hearing deficits are suspected, a brainstem auditory evoked response test should be performed. Authors’ address: The William R. Pritchard Veterinary Medical Teaching Hospital, University of California, Davis, CA 95616; e-mail: mraleman@ucdavis.edu. © 2014 AAEP.

1. Introduction
Hearing loss in horses has been seldom investigated and can be a cause of difficulty in training, decreased performance, and behavior alterations. Brainstem auditory evoked response (BAER) testing is a noninvasive, objective, and easy to perform diagnostic modality that evaluates the integrity of the auditory pathway. The objective of the study was to describe BAER findings, common signs, and causes of hearing loss in adult horses.

2. Materials and Methods
BAER records from the Clinical Neurophysiology Laboratory were reviewed from the years of 1982 to 2013. Medical records were reviewed, and horses were grouped under disease categories. Descriptive statistics included mean, standard deviation, and range.

3. Results
Fifty-seven of 76 horses that had a BAER had auditory deficits. There was no breed, gender, or age predisposition with the exception of American Paint horses diagnosed with congenital sensorineural deafness. Auditory loss was bilateral in 74% and unilateral in 26% of the horses. The most common causes of auditory loss included temporohyoid osteoarthropathy (35%), congenital sensorineural deafness (30%) associated to certain coat and eye color patterns, multifocal brain disease (23%), and otitis media/interna (7%).

4. Discussion
Temporohyoid osteoarthropathy, multifocal brain disease, and otitis are common causes of hearing loss. Hearing should be investigated in horses with extensive white color markings.

Acknowledgments

Conflict of Interest
The Authors declare no conflicts of interest.
Auditory Loss in Horses, Part II: Foals

Monica Aleman, MVZ Cert., PhD, DACVIM (Internal Medicine, Neurology)*; John E. Madigan, DVM, MS, DACVIM, DACAW; Colette Williams, PhD; and Terrell A. Holliday, DVM, PhD, DACVIM (Neurology)

Common neonatal diseases such as sepsis, ischemic hypoxia, neonatal isoerythrolysis, prematurity, and congenital anomalies can result or be associated with auditory loss in foals. Therefore, evaluation of hearing should be part of the physical and neurological examination in ill-neonatal foals. Authors’ address: The William R. Pritchard Veterinary Medical Teaching Hospital, University of California, Davis, CA 95616; e-mail: mraleman@ucdavis.edu. *Corresponding and presenting author. © 2014 AAEP.

1. Introduction
Auditory deficits occur in foals but are rarely recognized. In human medicine, several neonatal diseases are common causes of permanent hearing loss in infants. Descriptions of auditory loss in neonatal foals are limited to 3 foals with lethal white foal syndrome. The association of auditory loss with common neonatal diseases has not been studied. Therefore, the objective of the study was to investigate auditory deficits in ill-neonatal and non-neonatal foals.

2. Materials and Methods
Brainstem auditory evoked response (BAER; a study to investigate auditory function) records from the Clinical Neurophysiology Laboratory were reviewed from the years of 1982 to 2013. Clinical data were extracted from the medical records. Descriptive statistics were performed.

3. Results
Ten of 15 neonatal foals had absent BAER and were associated with sepsis, neonatal encephalopathy, neonatal isoerythrolysis, and prematurity. Auditory deficits were also observed in foals with certain coat and eye color patterns such as completely or mostly white with blue irises and lavender with pale yellowish irises. One non-neonatal foal (n = 1/3) with an intracranial abscess had auditory loss.

4. Discussion
Hearing deficits occur in foals and are associated with common neonatal diseases and certain coat and eye color patterns. Neurologic and intracranial disease can also result in auditory loss in foals of any age.

Acknowledgments
The Authors declare no conflicts of interest.
How to Manage Foal Rejection

Mary Stewart E. White, BS; and Charles F. Scoggin, DVM, MS, DACT*

Foal rejection is an uncommon but important problem that can adversely affect the well-being of equine neonates. Various methods are available for managing and overcoming foal rejection. These include physical and/or chemical restraint, behavior modification, and hormone therapy. Surrogation is utilized when a foal's dam experiences severe illness, death, or outright rejection of the foal, all of which can prevent her from raising her foal. Methods commonly used to treat foal rejection are also used to graft foals onto surrogate mares. Authors' address: Claiborne Farm, PO Box 150, Paris, KY 40362; e-mail: cscoggin@claibornefarm.com. *Corresponding and presenting author. © 2014 AAEP.

1. Introduction

While relatively uncommon, foal rejection is a serious condition that can adversely affect the health and welfare of an equine neonate. Consequences can be costly and labor intensive for the human caretakers and they can cause untoward and sometimes fatal injuries to the foal. Foal rejection can be seen in all breeds of horses, with the highest rate reported in Arabians (5.1%), followed by Paint Horses (1.9%) and Thoroughbreds (<1%).1 Another important consideration is that a mare that has previously rejected her foal is at an increased risk of doing so again.2

Normal Maternal Behavior

Most signs of normal maternal behavior are fairly easy to recognize. Nickering at and licking and nuzzling of the foal are commonly seen traits. A dam will shield her foal from humans and other horses and she will often stand over her foal while it is recumbent. More subtle signs include the mare stepping a hind leg back to offer greater exposure to the udder and gently nudging the foal as it is searching for a teat. These motherly instincts are thought to develop from various sensory, tactile, and mental stimuli. Stimulation can occur with olfactory recognition of fetal fluids and membranes, physical contact, nursing, and visual exchanges with the foal. These actions—along with other factors that might not be readily apparent—help foster normal attentive and protective maternal behavior.

Endogenous hormones are important for developing a female's instincts and ability to care for and nurture her offspring. The neuropeptide oxytocin seems to be important in developing proper maternal behavior. Oxytocin is synthesized in the hypothalamus and released from the posterior pituitary gland. While commonly thought of as an ecbolic agent whose primary target tissue is the myometrium of the uterus, oxytocin has been demonstrated to have effects within the central nervous system. Indeed, its importance in stimulating maternal behavior has been described in humans,3 lab animals,4 and farm animals.5,6 In addition, progesterone and estrogen may also play a role in the development of maternal behavior. For example, a preliminary study showed lower serum concentrations of these hormones in rejecting mares compared to nonreject-
ing mares.\textsuperscript{1} Lactation—an important milestone in the immediate postpartum period—is mediated through a multitude of hormones, including the aforementioned sex hormones and oxytocin, as well as growth hormone, prolactin, dopamine, and insulin-like growth factor 1. Consequently, knowledge of the hormonal events involved in maternal behavior, lactogenesis, and milk letdown can become important when identifying causes for and managing cases of foal rejection.

Abnormal Maternal Behavior and Foal Rejection

Abnormal maternal behavior and foal rejection can be perplexing and, at times, disturbing to witness. Mares may kick, squeal, or chase their foal, while others may simply seem indifferent to or become elusive from their foals. Different forms of foal rejection have been previously described by Crowell-Davis and Houpt.\textsuperscript{1,7} In our practice, we typically see two types of foal rejection. The first is aggressive rejection, which is typified by overt violence and hostility towards the foal. Signs include biting, kicking, and savage attacks. The second is avoidance rejection and is characterized by disinterest of the mare and/or evasion from the foal. For instance, the mare may walk away from the foal when it attempts to nurse or fail to display any interest in the foal, especially when the two are removed from eyesight of each other. A salient point regarding these classifications is that they are not mutually exclusive; indeed, variations of the two can occur depending upon the circumstances and individuals involved in each case.

In some cases, the cause for foal rejection is readily apparent. For example: the mare could be young and/or inexperienced; she could have previously rejected a foal; she could feel anxious or nervous around the foal due to a change in environment or management; she could have poor milk production secondary to a disease process; she could have produced an abnormal or sick foal; or the scent of the foal could have been altered through drugs, such as dimethyl sulfoxide (DMSO), which could interfere with olfactory recognition.\textsuperscript{2} All of these instances can disrupt bonding time or hamper the natural progression of motherly behavior, thereby leading to foal rejection.

Other times, the reasons for rejection are not so clear. The clinician must obtain a thorough history and perform a careful physical examination of the mare and foal to decide if there are any issues that may be contributing to the abnormal behavior. Watching the pair interact can be useful for determining certain behavioral cues that might be contributing to rejection. It also behooves the clinician to carefully evaluate the management practices of the farm to ascertain whether changes in the environment have led to maternal aggression or avoidance.

One factor that is sometimes overlooked, yet highly important, is that of maternal pain. Pain can be secondary to either normal physiologic processes or certain disease conditions. Examples of the former include pain arising from postpartum uterine contractions or discomfort from a sore udder due to an aggressively nursing foal.\textsuperscript{2} Examples of the latter include musculoskeletal injury, such as laminitis or osteoarthritis, and systemic illness, such as metritis associated with retained fetal membranes or gastroenterocolitis. Identifying the source of pain underscores the importance of performing a thorough physical examination, while proper management of the pain can result in a successful outcome for all parties involved.

The safety and health of the foal should be of primary concern when dealing with cases of foal rejection. As such, the foal should be immediately removed from a mare that displays overt aggression towards the foal. If necessary, the foal should be administered at least one pint of good quality colos- trum through a nasogastric tube for passive transfer of immunoglobulins and nourishment. Alternatively, or in addition, intravenous plasma can be used to provide adequate passive transfer. Subsequent feedings can be performed every two to three hours with the mare’s own milk (provided she is tractable for harvesting milk), goat’s milk, or a commercial foal milk replacer formula\textsuperscript{a} from a pan, bottle, or indwelling nasogastric feeding tube. Supportive care, in the form of intravenous fluids, and prophylactic administration of broad-spectrum antimicrobials may also be administered at the discretion of the clinician to prevent or treat dehydration and sepsis.

2. Materials and Methods

Strategies that can be employed to treat and manage foal rejection include physical restraint, behavior modification, pharmaceutical intervention, and surrogation. Depending on the circumstances and individuals involved, these strategies can be used alone or in conjunction with one another. Also, farm management practices and available resources may preclude the use of some of these strategies. The clinician must thus work in cooperation with the owner or caregivers to determine which strategies could be useful, as well as which ones are feasible with respect to their experience and wherewithal.

Success in establishing normal maternal behavior or adequate surrogation can be determined by using at least three subjective markers. The first marker is the mare’s response following removal of the foal from eyesight. If the mare whinnies, becomes anxious, or attempts to follow the foal, then these are good indicators of the mare’s acceptance of and bonding with the foal. The second marker is leaving the mare and foal free from restraint in a stall or small enclosed area and observing their interactions. Positive indicators include: witnessing the foal physically contacting the mare without the mare showing any signs of aggression or indiffer-
ence; seeing the mare willingly allow the foal to nurse; and observing the mare standing over the foal as it is lying down. The third positive marker is turning the pair out in a paddock and seeing them remain in close proximity to one another, with the mare permitting the foal to nurse her at its own volition.

Physical Restraint

Physical restraint is one of the most commonly used means of dealing with foal rejection. A mild method of restraint is that of having a handler hold the mare steady by her halter and a lead rope (Figs. 1 and 2). Doing so can prevent the mare from walking off, swinging her hind end away from, or turning towards the foal as it attempts to nurse, all of which can significantly impede the foal’s ability to seek the udder. This particular method seems to be highly effective for inexperienced or maiden mares in that it can condition them to the presence of the foal when in the region of the udder.

There are several other options for restraint. These include the use of stocks or a barricade (Fig. 3), nose twitch (Fig. 4), lip chain (Figs. 5 and 6), neck pinch or “shoulder roll” (Fig. 7), and breeding hobbles. These methods are often employed when the mare demonstrates signs of aggression towards the foal, especially kicking or biting. Selection is dependent upon the available resources, experience of the handlers, and disposition of the mare. In the authors’ practice, a barricade is routinely used that provides good protection to the foal but, at the same time, allows easy access to the udder. Nursing behavior can be controlled by the use of a sliding door to limit the number of suckling attempts (Figs. 8 and

![Fig. 1. Physical restraint of the mare with a halter and lead rope allows for the foal to nurse by minimizing the amount of movement by the mare.](image)

![Fig. 2. Physical restraint allows for controlled interaction between the mare and foal.](image)

![Fig. 3. While a set of stocks or barricade is not available in all situations, it is both a safe and functional tool for managing foal rejection.](image)

![Fig. 4. Physical restraint with a nose twitch.](image)
9). Doing so prevents the mare from developing a sore udder and protects the foal if the pair needs to be left unsupervised for a period of time. One obvious downside to this setup is that it does not allow the mare constant visual access to the foal, especially when it is attempting to nurse. It is also important to allow the mare time out of the barricade or stocks so that she does not become stiff or develop distal limb edema.

The reader is advised that these methods of restraint are only temporary measures. If used too frequently, mares may become resentful and develop a negative association with the foal and nursing behavior. What is more, caution must be exercised when using a twitch or hobbles because they can—if used too severely or for too long of a time period—put the mare, foal, and human handlers at risk for injury.

Behavior Modification

Behavior modification primarily involves the use of positive reinforcement. This method entails the handler offering encouragement in a soft and soothing voice when the mare demonstrates signs of good maternal behavior. Gentle rubbing along the neck or between the eyes can also be used to comfort the mare. Another option is to offer grain or treats as the foal nurses. Doing so presents a reward for good behavior and can, indirectly, create a mild distraction for the mare as the foal attempts to engage and nurse her. Negative reinforcement is less frequently used and is performed to dissuade a mare from acting aggressively towards her foal.
Oftentimes, behavior modification is used in conjunction with physical restraint. A good example is holding a mare via a lip chain as the foal attempts to connect with and nurse the mare. Pressure can be released on the chain when the mare demonstrates interest in the foal and a soothing voice and gentle rub along the neck can reward the mare for good behavior.

Pharmaceutical Intervention

Chemical restraint, through the use of tranquilizers and sedatives, is another option when dealing with mares that are actively rejecting their foals. The phenothiazine tranquilizer, acepromazine maleate\textsuperscript{b} (0.05 mg/kg, IV or IM, q 8–12 h), is advocated by some authors\textsuperscript{1} because it can provide both tranquilization and stimulate lactation via its antidopaminergic activity. Other agents, such as the \(\alpha_2\)-agonists, xylazine\textsuperscript{c} (0.5–1.1 mg/kg, IV, q 8–12 h) and detomidined (0.001–0.002 mg/kg, IV or IM, q 8–12 h), and the opioid agonist/antagonist, butorphanole\textsuperscript{c} (0.02–0.05 mg/kg, IV, q 8–12 h), may also be used either alone or in conjunction with acepromazine to provide tranquilization, sedation, and/or analgesia. Prudent use of tranquilizers and sedatives is important so as to prevent ataxia and ileus. Anecdotally, paradoxical reactions have been reported in horses administered xylazine whereby they can become aggressive and overly sensitive to external stimuli (i.e., the “xyla-mean” effect). Therefore, clinicians should be careful in their selection of and criteria for the use of tranquilizers and sedatives so as not to further compound problems associated with foal rejection.

Another means of pharmaceutical intervention is administration of a relatively high dose of prostaglandin-F\textsubscript{2\alpha} (PGF\textsubscript{2\alpha}). This technique was first described by Daels and is hypothesized to trigger oxytocin release in the brain to promote concaveation and normal maternal behavior.\textsuperscript{7} A hybridized protocol utilized by the current authors begins with removing the foal from eyesight of the mare and giving her a large dose of dinoprost tromethamine\textsuperscript{d} (0.033–0.056 mg/kg, IM). Approximately 15 to 20 minutes after receiving PGF\textsubscript{2\alpha}, mares will show signs of intense sweating and cramping, as well as stream milk. With one person holding the mare and another leading the foal, the foal is taken back in the stall and presented at the mare’s head. Often times, the mare will nuzzle and nicker at the foal. The foal is then directed towards the mare’s udder and encouraged to nurse. Within a relatively short time period (~15–30 minutes), the mare usually accepts the foal and the two can be turned loose and left alone to interact and strengthen their bond. This protocol seems to effectively “reprogram” the mare’s behavior to where she becomes highly attentive to and accepting of the foal. If the mare still rejects the foal after the first attempt, this method can be repeated 24 h later.

The preceding protocol is not without possible complications, most of which are often associated with the administration of exogenous prostaglandins in horses. These include signs of colic, diarrhea, and anxiety. Because a larger dose is used than what is normally given to induce luteolysis, these signs can be more intense but are usually transient and self-limiting. It should be noted that the likelihood and severity of adverse reactions seem to increase when multiple doses of PGF\textsubscript{2\alpha} are used over the course of several days, so it may be necessary to either scale back the dose of PGF\textsubscript{2\alpha} or simply utilize another means to get the mare to accept the foal. The synthetic PGF\textsubscript{2\alpha}-analog, cloprostenol\textsuperscript{e} (0.001–0.002 mg/kg, IM)\textsuperscript{e} can be used instead of dinoprost because it is thought to produce less severe side effects compared to dinoprost.

Administration of progestogens is another option to promote proper maternal behavior. Use of progesterone in oil\textsuperscript{b} (150 mg/horse IM q 24 h) and the synthetic progestogen, altrenogest\textsuperscript{f} (0.044 mg/kg PO q 24 h), have been recommended by some clinicians.\textsuperscript{1,2} These treatments are hypothesized to exert a calming effect, raise the threshold for aggression, promote motherly behavior, or a combination thereof.

Some mares may not produce sufficient amounts of milk to support and satiate their foals. They can subsequently become “teat sore” due to the foal constantly and aggressively attempting to nurse, which can cause mares to become aggravated and hostile towards their foals. In these instances—and as long as common causes of agalactia have been ruled out (e.g., metritis and fescue toxicosis)—treatment with nonsteroidal anti-inflammatory agents, warm compresses, and the dopamine antagonist, domperidone\textsuperscript{e} (1.1 mg/kg, PO, q 12–24 h), can be utilized to reduce inflammation and promote lactation. Oxytocin\textsuperscript{g} (5–20 IU, IM or IV, q 2–6 h) can be administered to promote milk letdown. Finally, improving the plane of nutrition can also be of benefit when attempting to improve milk quantity.

Medicine Potpourri: Neurology, Foals, Muscles, and the Gut

*Close this window to return to IVIS*
Surrogation

Surrogate mares are often utilized when a foal’s dam experiences severe illness or death. In these instances, one of two options exists. The first is to utilize a wet nurse mare from either a commercial supplier or another mare that has recently lost her foal. Commercial nurse mare operations are fairly prevalent in Central Kentucky but are scarce in other parts of the country. As such, the other option is to stimulate lactation in a barren mare. Preferably, the mare should have raised foals in the past, possess an even temperament, be tractable to handling and restraint, have good udder conformation, and be free of disease and musculoskeletal issues.

Methods to induce lactation in nonpregnant mares have been described previously.8,9 Lactation is stimulated with a combination treatment of progesterone and estrogen, as well as antidopaminergic agents, such as domperidone or sulpiride. Progesterone is responsible for promoting lobuloalveolar growth within the mammary gland, while estrogen stimulates cellular division and development of the mammary ducts. When combined with treatment with a dopamine antagonist, this protocol can be used successfully to promote lactogenesis in barren mares. The reader is directed to publications by Daels8 and Steiner9 for specific information regarding these protocols.

The actual grafting process utilizes many of the same previously described methods to counteract foal rejection. Moreover, since olfactory recognition plays an important role in stimulating normal maternal behavior, the authors find it useful to mask or alter the scent or smell of both the mare and foal. If a lactating mare is going to be used and fresh fetal fluids and membranes are available, they can be applied to the body of the foal and around the nostrils of the mare. If these are not available, or if the mare is barren, then a grafting spray can be used to disguise the smell of the foal. Muzzling the foal for one to two hours is commonly done prior to introducing it to the surrogate. A hungry foal will usually seek the udder immediately upon presentation to the mare.

3. Results

Several of the previously described techniques have been utilized in the authors’ practice for the past six years. During this time, we have treated eight mares (average age 5.6 years; range 4–6 years) that initially rejected their foals and required some form of intervention to make them accept their foals. Physical restraint alone was used in one case (12.5%), a combination of physical restraint with tranquilization was used in five of the eight cases (62.5%), and the high-dose PGF2α method was used in two of the eight cases (25%). In all instances, we achieved a 100% success rate in inducing proper maternal behavior and foal acceptance within 72 hours (average = 52.5 h; range = 12–72 h) of foaling.

We have also employed these techniques on a total of 24 lactating nurse mares (ages unknown) with 22 foals (range of 6 h– to 32 days of age). All but two mares accepted the foals within five days (average = 59.0 h; range = 2–120 h) of first introducing a foal to them, for a grafting success rate of 91.7%. Physical restraint in combination with tranquilization was all that was required in 10 of these mares (10/24 or 42.0%). The high-dose PGF2α method was used in the remaining 14 mares, and 12 of these mares (12/14 or 85.7%) adequately bonded with the foal within five days of being introduced to the foals. All of these mares were actively lactating, so exogenous progesterone and estrogen were not utilized in any of the mares. However, in three of the cases in which high-dose PGF2α was administered (3/14 or 21.4%), domperidone was administered for two to three days following grafting because these mares’ milk production was judged to be less than adequate.

4. Discussion

Foal rejection is a relatively uncommon occurrence in clinical equine practice. Nevertheless, when it does happen, it can be frustrating to deal with and unsettling to witness. Fortunately, several different options exist for treating and managing foal rejection, which range from physical restraint, to behavior modification, to pharmaceutical intervention. These methods can be used alone or in combination to fit each individual case or remain within the limits of available resources. We recommend starting with the simple methods (e.g., physical restraint with or without tranquilization) and then progressing to more involved techniques, such as behavior modification and high-dose PGF2α administration, if the circumstances dictate the use of more aggressive tactics. Our experience indicates that avoidance rejection is easier and quicker to reconcile compared to aggressive rejection. In many cases, light manual restraint with light to moderate sedation works well in mares that are avoiding but not aggressive towards their foals. Aggression oftentimes requires more forceful and involved measures, such as moderate restraint (e.g., nose twitch or lip chain), behavior modification, and high-dose PGF2α, but not necessarily in that order. Luckily, we have been able to successfully treat all of our foal rejection cases within 72 hours from the birth of the foal, which is a shorter period of time than the two to three weeks previously reported in the literature.1

With respect to surrogation, there are a couple of concluding thoughts. First, the age of the foal and time frame in which the surrogate had her foal seems to impact the time required for adequate bonding. If the foal is relatively young (e.g., less than 7 days) and the mare has recently foaled (e.g., 24–48 h), the grafting process is often very swift and seamless. Older foals or mares that have been raising a foal for several days appear to require more time and effort. However, with persistence and pa-
tience, success can often be achieved within five days of introduction. Second, the authors are of the opinion that surrogation is a much better option than attempting to hand-raise an orphan foal. Anecdotally, orphan foals seem to display unsafe and unusual behavioral traits towards humans and other horses as they mature. This phenomenon has been coined the “orphan foal” syndrome. It is likely a manifestation of overly intensive handling and rearing by humans, as well as inadequate socialization with other horses during an early yet highly formative time period. Foals raised by nurse mares seem—on average—more respectful towards humans and better adjusted to a herd environment compared to orphan foals. Finally, surrogation does not appear to hinder foals’ development or place them at a higher risk for disease relative to other foals. In the authors’ practice, the rate of growth and adverse health events appear to be equivocal between foals raised on a nurse mare and those raised by their own dams.

Acknowledgments

Conflict of Interest

The Authors declare no conflicts of interest.

References and Footnotes


*Foal-Lac Powder, Pet Ag, Hampshire, IL 60140.
*Aceproject, Henry Schein Animal Health, Dublin, OH 43017.
*sAnaSed®, Lloyd Laboratories, Shenandoah, IA 51601.
*dDormosedan®, Zoetis, Florham Park, NJ 07932.
*eTorbogesic®, Zoetis, Florham Park, NJ 07932.
*fLutalyse®, Zoetis, Florham Park, NJ 07932.
*gEstrumate®, Merck Animal Health, Millsboro, DE 19966.
*hProgesterone in oil, Rood and Riddle Veterinary Pharmacy, Lexington, KY 40511.
*iRegumate®, Merck Animal Health, Millsboro, DE 19966.
*jEquidone®, Dechra Veterinary Products, Overland Park, KS 66211.
*kOxoject, Henry Schein Animal Health, Columbus, OH 43204.
*lMotherUp®, Pierre, SD 57501.
How to Build an Automatic Milk Feeding Device for Orphan Foals

Jenni Schroeder, DVM

1. Introduction
Veterinarians often face the dilemma of having a newborn foal in their care that may require round the clock feedings of a supplemental milk source. Foals whose dams are deceased, have rejected them, or are simply not producing enough milk normally require either a nurse mare or an alternative supply of milk or milk replacer. Once a foal’s antibody requirements have been met with the ingestion of colostrum or the delivery of an IV hyperimmune plasma, a decision will need to be made regarding the source and delivery method of its nutrition. A foal will require some form of milk for the first 2 to 5 months of life.1 A commercially available nurse mare or an open mare who has been hormonally induced to lactate are two options that offer both a long-term source of naturally balanced nutrition and social upbringing. Unfortunately, both of these alternatives take valuable time to arrange, can be very costly, and still run the risk of foal rejection. In the meantime, it is necessary to provide feedings at 1–2 hour intervals for at least the first few days of the foal’s life.2 Owners may quickly become exhausted by feeding foals this frequently and paying staff can rapidly become cost prohibitive. Some caretakers are tempted to leave out large volumes of milk in the stall overnight for the foal to consume at will. This practice can lead to spoilage of the milk or overconsumption by the foal, which can cause colic, diarrhea, gastric ulceration, and lack of weight gain.3 Constructing a relatively inexpensive automated device to store, refrigerate, and distribute a programmed volume of milk into a small receptacle for consumption at timed intervals can save the practitioner or owner time, money, and provide a more natural digestive process for the foal.

2. Materials and Methods
An area with a 110 V electrical outlet and protection from rain is required to run and maintain the feeding device. Always use caution to avoid flammable materials around any electrical device, and be sure the device does not sit in wet conditions. The materials required are readily available online, and overnight shipping can be used to expedite delivery. A busy breeding farm or veterinary hospital can construct the device before the beginning of foaling season to ensure it will be ready when needed. The unit as described here costs less than $400 in supplies to build, and our hospital was quickly able to reimburse costs by charging a deposit and monthly rental fee for at home use. Equipment to be purchased includes the following:

1. A compact (dormitory) refrigerator. We use a Kenmore Chill 3.3 cu. ft. model no. 93382.
which averages $143.99 at www.sears.com, $129.99 at www.amazon.com, or is available at Sears appliance stores. This fridge allows a larger milk receptacle to be used inside, as some compact refrigerators do not provide a large enough ventral footprint on which to place a container large enough to store milk for 12 hours of use.

2. A rectangular milk storage container that fits inside the chosen compact refrigerator. We use a 2.5 gallon Sterilite\textsuperscript{b} plastic trash can found at Dollar General or Walmart for around $2.00.

3. A peristaltic dosing pump with a timer. These can be purchased separately as a pump and programmable electric timer or as one unit. We have tried several methods with different pumps and timer combinations, and the listed all in one programmable peristaltic pump seems to be the most affordable for what it provides. We use a 115 VAC Beta DR-2000 Clock Based Chemical Feed System\textsuperscript{c}. This unit can be purchased at www.cannon-water.com for $224.50 at the time of publication although they can occasionally be found new or used on www.ebay.com for less. This pump uses a standard 110 V outlet and pumps an average of 150 mL/min of fluid. It can be programmed to cycle once an hour for up to 20 minutes, which would provide a 3 L maximum output per cycle. It may be programmed to cycle more frequently than once an hour but only stores 24 programmed cycles to memory, so the internal clock would need to be reset every 12 hours for more frequent feeds. The pump is very easy to program once you read the enclosed instructions. Using a peristaltic type pump keeps the milk out of the moving pump parts to avoid contamination. Peristaltic pumps usually require very little maintenance or part replacement. The pump tube inside the roller assembly may need replacement every few years depending on how well it is cleaned and the conditions of storage. All the major components can be purchased from Cannon Water. The internal battery of this particular pump will only store programming for 6 months when unplugged, so if it is stored unplugged for more than 6 months in the offseason, then it will have to be reprogrammed at the beginning of each season and a new replacement battery may be purchased from the distributor. When purchased from the distributor, the pump is supplied with the remainder of the fittings including ¼ inch tubing, a mounting bracket, screws, drywall anchors, cable ties, squeeze tube fittings, and standpipe to stiffen the suction tubing and help keep it near the bottom of the milk receptacle.

4. An optional clear storage container to protect the pump from water and dust in a barn type environment. We used a 4 L plastic food storage container found at the Dollar Store or Walmart for $2.00 to $6.00.

An electric drill, a box blade, a 3/16 inch drill bit, a ¼ inch drill bit, a marker, a hammer, and a Phillips head screwdriver are tools used to assemble the device.

Unpack the compact refrigerator and ensure the chosen milk receptacle fits within the closed refrigerator. Choose whether to mount the peristaltic pump (Fig. 1) to the top or the side of the refrigerator. The pump may be mounted with a sliding bracket or with 3 screws (Fig. 2). In this example, we have mounted the pump to the top of the refrigerator with 3 screws. If the pump is mounted within a protective container, ensure the pump fits within the closed container and use the box blade to cut exit holes for the power cord and intake and output tubing (Fig. 3). Place the container and pump in the selected mounting position on the top or side of the refrigerator, and use the marker to mark the position of the suction hose entrance through the side of the refrigerator at a level just
below the freezer unit (Fig. 7). Once marked, use the ¼ inch drill bit to drill a hole through the thin metal housing and foam insulation on the side of the refrigerator. Cut the desired length of suction tubing to run between the pump and the bottom of the milk receptacle. Feed a section of suction tubing through this hole and to the bottom of the receptacle. Attach the stiff standpipe tubing provided with the pump to the distal end of the suction tube and place within the milk receptacle so it touches the bottom (Fig. 8). Attach the proximal end of the tube to the intake (suction) side of the peristaltic pump using the provided squeeze tube fittings. Silicone may be used to seal the exit hole through the wall of the refrigerator if desired. Attach the remaining section of tubing to the output port on the pump (Fig. 9). Run the section of output tubing to distribute milk into a chosen receptacle in the stall from which the foal is to drink. Use cable ties as needed to fasten the tubing in place.

Most foals can quickly be taught to drink from a bucket by placing a milk coated finger in their mouth and while they are suckling, slowly inserting...
the finger into a shallow pan or bucket leading their mouth to the source of milk. For foals that drink well from a bottle but have difficulty learning to drink from a bucket, a Calf-Mate calf nursing bucket\texttrademark can be purchased at www.eNasco.com and modified by cutting the end of a smaller human or goat nipple to fit the output.

Several commercially available foal specific milk replacers can be ordered online or found at local feed stores. The author’s hospital most commonly uses Mares Match\textsuperscript{a} by Land O’Lakes or Foals First\textsuperscript{b} by Progressive Nutrition, as we have had more success with palatability and less incidence of diarrhea with these brands. Follow the packaged instructions carefully for preparation of the product. Some choose to dilute the foal replacer to half strength for

Fig. 6. Place the pump inside the container, and use the Phillips screwdriver to screw in the mounting screws provided with the pump.

Fig. 7. Mark the area for milk tube placement through the side of the refrigerator just below the freezer coil shelf. Use the ¼ inch drill bit to drill a hole through the side of the refrigerator.

Fig. 8. Measure and cut the required length of tubing to run between the input port on the pump, through a hole drilled into the side of the refrigerator, and down to the bottom of the chosen milk receptacle. Attach standpipe to bottom of tubing and place at bottom of receptacle.

Fig. 9. Attach the remaining tubing to the output port of the pump. This will be used to run into the bucket where the foal is housed and secured with cable ties.
the first few feedings to acclimate the foal to taste if it was originally nursing from a mare and to avoid constipation. Avoid any sudden change in brands of milk replacer. The average foal requires 10 to 15% of its body weight in milk the first few feedings and then 25 to 35% of its body weight after that until it gradually begins to be able to digest disaccharides and fiber between 2 and 4 months when the Maltase and Sucrase enzymes increase in levels and cecal microflora are established. The weight of milk is approximately 1 kg/1 L so a 50 kg foal will need at least 5 L a day in the first few days and gradually be increased to 12.5 to 15 L as it grows. The average foal will gain 2 to 3 pounds a day. The timer on the machine is set to dispense the desired amount of milk per hour. We begin by setting the timer at 2 minutes per hour, which will dispense an approximated 300 mL an hour or 7.2 L a day. The chosen receptacle needs to be small enough, placed at the proper height, and possibly placed at a slight angle so that the foal's muzzle can fit to the bottom and drink the entire amount. As the foal grows, the timer is changed to pump for longer time periods distributing larger volumes of milk and at decreased intervals. The pump can be used until it is possible to get down to a more owner manageable 5 times a day feeding at around 6 weeks old or used for the first several months until it is eating a normal diet. Once the pump is done being used for a particular foal, 10% bleach water is pumped through the pump tubing followed by distilled water and then air to disinfect and store.

3. Results
The described device has worked well in our hospital setting, and as previously described, we often send them home with orphan foals for a monthly rental fee or more distant clients chose to purchase the device. We have used this automated method for over 20 orphan foals in our practice. The foals quickly learn the sound of the pump when it is activated and run to the bucket to drink. The foals gained weight at an average pace and no significant diarrhea, colic, or constipation was encountered. The owners gave positive reviews regarding the functionality and convenience of the device when it was returned for a deposit refund. We did have an instance in which the tubing required replacement due to damage by mice trying to reach the milk inside. The programmable pump was programmed as needed to gradually increase the volume and decrease the frequency of feedings as the foal grew and the intestines adapted to the introduction of roughage and grains.

4. Discussion
Having a programmable automated device available to offer allocated amounts of milk replacer to an orphaned foal can be a very convenient time and money saving option. Additionally, it can mimic a more natural feeding schedule. This can help avoid overfilling of the stomach, which can lead to delayed gastric emptying and proliferation of enteric pathogens. More frequent meals can decrease the incidence of gastric ulceration as well as maintain glucose levels and gut motility in the foal. The foals will also avoid being exposed to frequent human bonding as in a bottle feeding situation, which can lead to later behavioral problems. Presenting the option of a relatively inexpensive automatic feeding device is a good way to offer clients some peace of mind in a stressful situation. Although their mare may have passed away or rejected her foal, they do not need to worry about the foal's growth potential. Owners that typically visit the barn twice daily for morning and evening feeds have the ability to program and fill the device at these times without missing work or hiring extra staff to feed an orphaned foal. Veterinary hospitals without 24 hour staff can also benefit from the availability of this device for overnight feedings.

Acknowledgments

Conflict of Interest
The Author declares no conflicts of interest.

References and Footnotes

*aKenmore 3.3 cu.ft. Compact Refrigerator, Kenmore, Hoffman Estates, IL 60169.
*bSterilite Corporation, Townsend, MA 01469.
*cCannon Water Technology Inc., Rocklin, CA 95765.
*dCalf-Mate Calf Feeder, Fortiflex, Miami, OK 74354.
*eMares Match foal milk replacer, Land O Lakes Animal Milk Products Co., Shoreview, MN 55126.
*fFoals First Milk Replacer Powder, Progressive Nutrition LLC, Brookville, OH 45309.
Frequency of Ultrasonographic Visualization of Liver in Published Sites for Blind Biopsy in Healthy Horses

Sara C. Sammons, DVM, MS*; Tracy E. Norman, VMD, DACVIM; M. Keith Chaffin, DVM, MS, DACVIM; and Noah D. Cohen, VMD, MPH, PhD, DACVIM

The practice of blind liver biopsy in the published recommended locations in horses carries significant potential risks. Ultrasound-assisted liver biopsy is recommended to reduce the risk of serious complications. Authors' address: Department of Large Animal Clinical Sciences, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University, College Station, TX 77843; e-mail: SSammons@cvm.tamu.edu. *Corresponding and presenting author. © 2014 AAEP.

1. Introduction
Percutaneous liver biopsy is regarded as the best antemortem test for equine hepatopathy. Blind liver biopsies are still performed by practitioners, despite potential associated risks such as pneumothorax or hemorrhage. Direct or indirect ultrasound guidance allows for identification of the most appropriate site for liver biopsy. Our goal was to identify the frequency of sonographic identification of adequate liver tissue for biopsy in the published blind liver biopsy locations.

2. Materials and Methods
Serum liver parameters were evaluated in 36 middle-aged Quarter Horses to ensure that only horses with normal liver function were included in the study. The published blind liver biopsy region, just below a line drawn between the dorsal aspect of the tuber coxae and the point of the elbow in the right 11th to 14th intercostal spaces, was examined sonographically.

3. Results and Discussion
Only 39% of horses had liver visualized in the published blind liver biopsy region. None of the 36 horses had liver of adequate thickness for biopsy in the published region. When liver was not visible, lung was visualized instead in 55% of horses, bowel in 36% of horses, and both bowel and lung in 9% of horses. In many horses, liver tissue may be inadequate for biopsy in the published blind biopsy locations.

Acknowledgments
Supported by a grant from the Department of Veterinary Large Animal Clinical Sciences, College of Veterinary Medicine and Biomedical Sciences, Texas A&M University.

Conflict of Interest
The Authors declare no conflicts of interest.
Owner-Reported Response to Treatment of 130 Headshaking Horses

Kirstie J. Pickles, BVMS, MSc, PhD, CertEIM, DECEIM; Monica Aleman, MVZ Cert, PhD, DACVIM†; David J. Marlin, BSc(Hons), PhD; Vicki J. Adams, BSc, DVM, MSc, PhD, MRCVS; and John Madigan, DVM, MS, DACVIM, DACAW*

Nose nets, face masks, and magnesium supplementation were the most efficacious nonmedical therapies for treatment of headshaking. Of the pharmacological treatments, cyproheptadine, corticosteroids, and melatonin resulted in greatest improvement. Each of these treatments resulted in improvement in approximately 50% of headshaking horses. Authors’ addresses: Department of Veterinary Medicine and Epidemiology, University of California Davis, CA 95616 (Pickles, Aleman, Madigan); Science Supplements, Unit 2A, Chase Road, Bury St. Edmunds, Suffolk, IP32 6NT, UK (Marlin); and PO Box 80, Bury St. Edmunds, Suffolk UK IP28 9BF, UK (Adams); e-mail: jemadigan@ucdavis.edu. *Corresponding author; †Presenting author. © 2014 AAEP.

1. Introduction

Idiopathic headshaking has been recognized for over a hundred years1,2 and is a spontaneously occurring disorder of mature horses causing violent head flicking, snorting, and muzzle rubbing. Due to this localization of clinical signs to the head, and in particular muzzle area, it has long been suspected that the disease pathogenesis involves the trigeminal nerve.1,3,4 Recently a decreased threshold for trigeminal nerve activation has been confirmed in headshaking horses compared to controls.5 The limited pathological studies performed to date have failed to determine any structural abnormalities of the trigeminal nerve, and therefore a functional disorder is proposed.5,6 Other unexplained features of headshaking include the predisposition of geldings to the disease and the seasonality of clinical signs.7,8 This seasonality and the fact that some horses go into spontaneous remission suggests that the documented aberrant activity of the trigeminal nerve may be reversible. However, due to our limited understanding of the etiopathogenesis of headshaking, current treatments are primarily directed at minimizing clinical signs and discomfort rather than correcting anomalous trigeminal neurophysiology and, consequently, have limited efficacy in many horses.4,9 There are few large scale studies reporting success rates of the various treatments for headshaking, which makes it difficult for veterinarians to provide such advice to owners. The aim of this study was to undertake a survey of owners of headshaking horses to determine the reported success rate of commonly utilized therapies.
2. Materials and Methods

Questionnaires

An online questionnaire was designed using Survey Monkey®. The questionnaire contained 3 sections. The first section comprised 27 questions regarding the horse’s signalment and general management. Section 2 contained 15 questions regarding the horse’s headshaking including if the horse was currently headshaking, which months of the year headshaking occurred, which month headshaking was worst, evidence of seasonality and weather related headshaking behavior, and if headshaking had been diagnosed by a veterinarian. Additionally, owners were asked to complete a checklist of 20 particular headshaking behaviors for their horse and a 15 item checklist about activities associated with their horse’s headshaking. Section 3 comprised 11 subsections regarding 11 possible headshaking treatments: nose net, face mask, fly control, cyproheptadine, carbamazepine, corticosteroids, non-steroidal anti-inflammatories (NSAIDs), antihistamines, melatonin, magnesium, and combined melatonin and magnesium therapy. If a particular treatment had not been used, the respondent was directed to the next treatment subsection. Within each treatment subsection, owners were asked to specify what time of year the treatment had been trialed, describe the particular treatment (e.g., type of nose net) and dosage and administration details if appropriate (including if product was generic or compounded), if any response was observed (completely stopped headshaking, marked improvement, some improvement, no change, or worsened headshaking), and if any side effects of treatment were noted. The questionnaire was advertised via headshaking website forums and via Facebook®. Links to the questionnaire remained open for 2 months.

Data Analysis

Results are reported descriptively. A positive treatment outcome was deemed as improvement in headshaking behavior and was calculated as the sum of completely stopped headshaking, marked improvement, and some improvement responses. A negative treatment outcome was described as no improvement or worsening of headshaking and was calculated as the sum of no change and worsened headshaking responses. For geographical comparisons, data was assigned a location of North America, Europe, or Australasia. Mann Whitney tests were used to examine 2 non-paired variables.

3. Results

Questionnaire Response

Questionnaires were completed by 137 respondents. The treatment section was not completed by 7 respondents giving 130 useable questionnaires. Some respondents gave incomplete details for a particular treatment giving a useable response range of 122 to 130 (median 127) for individual treatments. Responses were received from the UK (n = 49), U.S. (n = 45), Canada (n = 8), mainland Europe (n = 11, 3 each from France and Germany and 2 from The Netherlands), Australia (n = 3), and New Zealand (n = 5).

<table>
<thead>
<tr>
<th>Clinical sign</th>
<th>Prevalence (%)</th>
</tr>
</thead>
<tbody>
<tr>
<td>Shaking or flipping the head in a vertical plane</td>
<td>121 (92)</td>
</tr>
<tr>
<td>Acting as if an insect had flown up the nose</td>
<td>112 (85)</td>
</tr>
<tr>
<td>Rubbing the nose/muzzle on objects</td>
<td>108 (82)</td>
</tr>
<tr>
<td>Snorting</td>
<td>92 (62)</td>
</tr>
<tr>
<td>Rubbing nose/muzzle on ground while moving</td>
<td>81 (61)</td>
</tr>
<tr>
<td>Striking at nose/muzzle with front foot</td>
<td>70 (53)</td>
</tr>
<tr>
<td>Anxious expression</td>
<td>59 (45)</td>
</tr>
<tr>
<td>Headshaking reduced at night</td>
<td>59 (45)</td>
</tr>
<tr>
<td>Stumbling</td>
<td>42 (32)</td>
</tr>
<tr>
<td>Staring into space</td>
<td>38 (29)</td>
</tr>
<tr>
<td>Horizontal headshaking</td>
<td>37 (28)</td>
</tr>
<tr>
<td>Reluctance to move</td>
<td>36 (27)</td>
</tr>
<tr>
<td>Panic following staring episodes</td>
<td>32 (24)</td>
</tr>
<tr>
<td>Avoids light</td>
<td>32 (24)</td>
</tr>
<tr>
<td>Seeks shade</td>
<td>32 (24)</td>
</tr>
<tr>
<td>Licking or smacking of lips or excessive opening and closing of mouth</td>
<td>28 (21)</td>
</tr>
<tr>
<td>Worse when sunny</td>
<td>33 (25)</td>
</tr>
<tr>
<td>Worse when windy</td>
<td>29 (22)</td>
</tr>
<tr>
<td>Worse when rainy</td>
<td>13 (10)</td>
</tr>
<tr>
<td>Worse when humid</td>
<td>7 (5)</td>
</tr>
<tr>
<td>Better when cloudy</td>
<td>14 (11)</td>
</tr>
<tr>
<td>Better when rainy</td>
<td>13 (10)</td>
</tr>
</tbody>
</table>

Description of Headshaking Horses

The median (range) age of the population of headshaking horses was 11 (2–27) years with a median (range) length of ownership of 6 (0–20) years. Most breeds were represented with Thoroughbred (including crosses) and Quarter Horse being most frequent comprising 22% and 14% of the population, respectively. Almost half of the horses were used for light activities only with 24% used for hacking/trail riding, 16% for pleasure riding, and 8% being retired/companion animals. Dressage was the most frequent competitive discipline performed by headshakers, reported by 13% of owners.

Of the 132 horses for which data were available, 84 (64%) had been diagnosed with idiopathic headshaking by a veterinarian. Headshaking behavior was seasonal in 79/132 (60%) horses whilst 53/132 (40%) displayed clinical signs all year or randomly intermittently, without any apparent predictability. The most common headshaking signs displayed were “vertical shaking of the head” (121/132; 92%), “acting as if an insect flew up its nose” (112/132;
Cyproheptadine was compounded or not.

move (P = 0.01), when ridden at canter (P = 0.003), when walked in hand in a bridle (P = 0.04) or halter (P = 0.02), and when eating grass (P = 0.05).

For the 131 owners that gave data, 9 (7%) described their horse’s headshaking as mild (occasional headshaking, rarely interferes with riding activity), 35 (27%) as moderate (some headshaking, interferes somewhat with riding activity), 41 (31%) as severe (frequent headshaking, interferes greatly with riding activity), and 46 (35%) as very severe (frequent headshaking, impossible to perform riding activity). Owners of horses with a veterinary diagnosis rated their horse’s headshaking as more severe than owner-diagnosed horses (P = 0.007).

Treatment Outcome

Nose Net

Nose nets had been used by 110/128 (86%) respondents. Of those that had tried nose nets, 91 (83%) had used half nets extending from the noseband to cover the upper jaw and lip, 43 (39%) had used full nose nets covering both the upper and lower jaws and lips below the noseband, and 24 (22%) had tried both types of nose nets. A further 4 owners had used homemade nose nets made from nylon stockings, 1 used ropes dangling from the noseband and 1 used a dangling fringe from the browband.

A positive outcome was reported by 58/110 (53%) owners; 6 horses (5%) completely stopped headshaking, 21 (19%) showed marked improvement, and 31 (28%) showed some improvement (Table 2). Any improvement was seen immediately upon placement of the nose net although some horses required several sessions of wearing the net to accommodate its use. Six respondents said that the positive effect of the nose net had waned over time. A negative outcome was reported by 52 (47%) of owners with 40 horses (36%) showing no response and 12 horses (11%) headshaking worse with use of a nose net (Table 1). Negative side effects were shown by 32 horses (29%) of horses using nose nets with irritation by the net (24 horses) and panic (5 horses) described most frequently. There was no significant difference between half or full nets in outcome or the presence of adverse effects.

Face Mask

Face masks had been used as a treatment on 83/130 (64%) headshaking horses with 42 (51%) of these masks containing UV eye shades. Forty-four (53%) horses had a positive outcome from face mask use with 3 (4%) horses completely stopping headshaking, 13 (16%) showing marked improvement, and 28 (34%) showing some improvement (Table 2). A negative outcome was observed in 39 (47%) horses with 32 (39%) showing no response and 7 (8%) horses becoming worse following face mask use (Table 2). Negative side effects were noted in 18 (22%) horses with “spookiness,” tripping due to diminished vision and worsening of headshaking most frequently cited. The presence of UV eye shades did not affect outcome or occurrence of adverse effects.

Table 2. Owner Reported Response of 130 Headshaking Horses to Various Treatments

<table>
<thead>
<tr>
<th>Treatment</th>
<th>Used</th>
<th>Positive outcome</th>
<th>Completely stopped</th>
<th>Marked improvement</th>
<th>Some improvement</th>
<th>Negative outcome</th>
<th>No change</th>
<th>Worse</th>
<th>Side effects</th>
</tr>
</thead>
<tbody>
<tr>
<td>Nose net (n = 128)</td>
<td>110 (86%)</td>
<td>58 (53%)</td>
<td>6 (5%)</td>
<td>21 (19%)</td>
<td>31 (28%)</td>
<td>52 (47%)</td>
<td>40 (36%)</td>
<td>12 (11%)</td>
<td>32 (29%)</td>
</tr>
<tr>
<td>Face mask (n = 130)</td>
<td>83 (64%)</td>
<td>44 (35%)</td>
<td>4 (4%)</td>
<td>13 (16%)</td>
<td>28 (34%)</td>
<td>39 (47%)</td>
<td>32 (39%)</td>
<td>7 (8%)</td>
<td>18 (22%)</td>
</tr>
<tr>
<td>Fly control (n = 126)</td>
<td>84 (67%)</td>
<td>22 (18%)</td>
<td>3 (4%)</td>
<td>3 (10%)</td>
<td>15 (18%)</td>
<td>66 (79%)</td>
<td>66 (79%)</td>
<td>0</td>
<td>2 (2%)</td>
</tr>
<tr>
<td>Cyproheptadine* (n = 128)</td>
<td>29 (23%)</td>
<td>14 (48%)</td>
<td>4 (14%)</td>
<td>3 (10%)</td>
<td>7 (24%)</td>
<td>15 (52%)</td>
<td>13 (45%)</td>
<td>2 (7%)</td>
<td>14 (48%)</td>
</tr>
<tr>
<td>Carbamazepine (n = 125)</td>
<td>8 (6%)</td>
<td>2 (25%)</td>
<td>0</td>
<td>0</td>
<td>2 (25%)</td>
<td>6 (75%)</td>
<td>6 (75%)</td>
<td>0</td>
<td>3 (38%)</td>
</tr>
<tr>
<td>Corticosteroids (n = 127)</td>
<td>31 (24%)</td>
<td>17 (55%)</td>
<td>1 (3%)</td>
<td>7 (23%)</td>
<td>9 (29%)</td>
<td>14 (45%)</td>
<td>13 (42%)</td>
<td>1 (3%)</td>
<td>3 (10%)</td>
</tr>
<tr>
<td>NSAIDs (n = 126)</td>
<td>22 (17%)</td>
<td>4 (18%)</td>
<td>0</td>
<td>1 (5%)</td>
<td>3 (14%)</td>
<td>18 (82%)</td>
<td>18 (82%)</td>
<td>0</td>
<td>1 (5%)</td>
</tr>
<tr>
<td>Antihistamines (n = 122)</td>
<td>26 (20%)</td>
<td>12 (33%)</td>
<td>1 (3%)</td>
<td>4 (11%)</td>
<td>7 (19%)</td>
<td>24 (67%)</td>
<td>23 (64%)</td>
<td>1 (3%)</td>
<td>7 (19%)</td>
</tr>
<tr>
<td>Melatonin (n = 128)</td>
<td>17 (13%)</td>
<td>8 (47%)</td>
<td>0</td>
<td>1 (6%)</td>
<td>7 (41%)</td>
<td>9 (53%)</td>
<td>9 (53%)</td>
<td>0</td>
<td>7 (41%)</td>
</tr>
<tr>
<td>Magnesium (n = 128)</td>
<td>58 (45%)</td>
<td>25 (43%)</td>
<td>1 (2%)</td>
<td>11 (19%)</td>
<td>13 (22%)</td>
<td>33 (57%)</td>
<td>33 (57%)</td>
<td>0</td>
<td>4 (7%)</td>
</tr>
<tr>
<td>Melatonin and magnesium</td>
<td>11 (9%)</td>
<td>6 (55%)</td>
<td>0</td>
<td>1 (9%)</td>
<td>5 (45%)</td>
<td>5 (45%)</td>
<td>5 (45%)</td>
<td>0</td>
<td>4 (36%)</td>
</tr>
</tbody>
</table>

NSAIDs = non-steroidal anti-inflammatories; n = number of responses.

*Fifteen horses received compounded cyproheptadine, 12 horses received generic cyproheptadine, and 1 owner was unsure if cyproheptadine was compounded or not.
**Insect/Fly Control**

Insect control had been used by 84/126 (67%) of owners in an attempt to decrease headshaking. Of these, 18 (21%) reported a positive outcome, with 3 (4%) horses showing marked improvement and 15 (18%) some improvement immediately upon use (Table 2). No horses displayed increased headshaking following insect control measures. Two horses (2%) had negative side effects (skin irritation) following treatment.

**Cyproheptadine**

Cyproheptadine treatment had been used by 29/128 (23%) of owners to control their horses’ headshaking. Fifteen horses received compounded cyproheptadine, 12 horses received generic cyproheptadine, and 1 owner did not know if cyproheptadine was compounded. Six owners reported administering 0.3 mg/kg, 2 owners stated 0.1 mg/kg, 15 owners did not know the administered dose, and dosage was unable to be determined from information given by 6 owners using compounded product. Nineteen owners reported administering cyproheptadine twice daily, 2 owners once daily, and 8 owners did not report frequency of dosing.

Fourteen horses (48%) had a positive outcome with cyproheptadine treatment: 4 (14%) stopped headshaking completely, 3 (10%) showed marked improvement, and 7 (24%) showed some improvement (Table 2). Fifteen horses (52%) had a negative outcome with 13 (45%) having no change and 2 (7%) having worsening of headshaking. The median (range) time to observe a positive response occurred 3 (2–14) days following initiation of therapy. Additionally, 1 owner reported that clinical signs resumed within 2 days of cyproheptadine withdrawal. Three owners described a transient effect such that headshaking recurred during administration; 2 of these horses received generic cyproheptadine and 1 received compounded product. One additional owner commented that cyproheptadine had stopped working when changed to compounded product. Adverse effects were reported in 14 (48%) of horses receiving cyproheptadine including lethargy, drowsiness, incoordination, colic, and irritability.

**Carbamazepine**

Carbamazepine therapy had been tried by 8/125 owners (6%), 2 of which reported some improvement and the remaining 6 reported no change in headshaking (Table 2). Improvement occurred within several weeks in 1 horse and was not reported for the other improved headshaker. One owner reported administration of 5 mg/kg 3 times daily, 6 owners did not know the dosage administered, and the dosage could not be extrapolated from the information given by 2 respondents. Five of these horses with an unknown dosage received carbamazepine twice daily and 1 three times daily. Adverse effects of lethargy and drowsiness were reported in 3 horses.

**Corticosteroids**

Corticosteroid therapy had been given to 31/127 (31%) of headshakers. Of these, 9 received oral steroids, 12 were administered injectable steroids, 6 were given dexamethasone pulse therapy (DPT), and 2 horses received inhalational medication. A positive outcome was reported in 17/31 (55%) of horses of which 1 completely stopped headshaking, 7 (23%) showed marked improvement, and 9 (29%) had some improvement (Table 2). Improvement was most frequently described by owners (7/12) as occurring a few days after treatment commenced. Three owners reported a transient improvement only. A negative outcome was noted in 14 (45%) of horses with 13 (42%) having no change and one becoming worse following therapy. Adverse effects were observed in 3 horses (10%) and comprised 1 each of increased appetite, lethargy, and mania (as described by owner).

**NSAIDs**

NSAID therapy had been used by 22/126 (17%) of owners for treatment of their horses’ headshaking. Phenylbutazone was listed as the most frequently administered NSAID (13/18 responses). Four horses (18%) improved with NSAID therapy with 1 horse showing marked improvement and 3 horses (14%) showing some improvement whilst the remaining 18 horses (82%) showed no change in headshaking (Table 2). Hives were listed as an adverse effect of treatment in one horse.

**Antihistamines**

Antihistamine treatment had been used in 36/122 (30%) of headshaking horses. Most frequently used drugs were chlorpheniramine (9 horses), hydroxyzine hydrochloride (8 horses), loratadine or desloratadine (8 horses), diphenhydramine (7 horses), and pseudoephedrine hydrochloride pyrilamine maleate (3 horses). A positive outcome was described within a few days to a few weeks in 12 (33%) of horses with 1 horse completely ceasing headshaking, 4 horses (11%) showing marked improvement, and 7 horses (19%) mildly improving (Table 2). A negative outcome was seen in 24 (67%) horses with no change in 23 (64%) and increased headshaking in 1 horse. Adverse effects were observed in 7 horses (19%) with drowsiness cited most frequently.

**Melatonin**

Melatonin had been administered to 17/128 (13%) of horses, all of which were located in the U.S. except one in Canada and one in the Southern hemisphere. Dose range was 12 to 20 mg with 15 mg once daily cited most frequently (6 responses). Ten respondents reported giving melatonin at 5 PM/dusk, 1 administered melatonin in the morning, and 1 gave melatonin twice daily. Six horses started melatonin therapy in winter, 6 in spring, and 5 in the...
summer. Eight horses (47%) responded positively to melatonin within 1 to 4 weeks of therapy; marked improvement was seen in 1 horse (the horse given melatonin in the morning) and some improvement in the remaining 7 (including the horse given melatonin twice daily) (Table 2). No change in headshaking was reported in 9 (53%) of horses. Seven horses (41%) had a longer hair coat or did not shed their winter coat whilst on melatonin.

**Magnesium**

Dietary supplementation with magnesium had been used by 58/128 (45%) of owners as a headshaking treatment. Magnesium oxide was the most common formulation reported (26 responses) followed by malate (7 responses). Doses of 5 to 40 g were reported. A positive outcome was reported in 25 horses (43%) within 2 days to 4 weeks of initiating therapy; additionally 1 owner reported headshaking worsened within a couple of days of stopping magnesium supplementation. One horse completely stopped headshaking, 11 (19%) showed marked improvement, and 13 (22%) showed mild improvement (Table 2). A negative outcome was noted in 33 horses (57%) in which no change in headshaking was observed. Two owners reported that their horse seemed calmer whilst on magnesium, whereas 1 owner each reported nervousness and diarrhea as adverse effects.

**Melatonin and Magnesium**

Only 11/122 (9%) owners had used melatonin and magnesium combination therapy to treat their horses’ headshaking. Six horses (55%) responded positively with 5 horses (45%) showing some improvement and 1 horse (10%) marked improvement in headshaking whereas 5 horses (45%) had no change (Table 2). All respondents who gave data (n = 7) used a dosage of 15 to 20 mg melatonin (median 15 mg) at 5 PM and 5 to 40 g magnesium. Two owners initiated combination therapy in spring, 5 in summer, and 4 in winter. Adverse effects were observed in 4 horses (36%), 3 of which did not shed their winter coat and 1 which gained weight and became lethargic approximately 1 month after therapy was initiated.

### 4. Discussion

Treatment of headshaking horses is challenging as therapies are largely directed towards managing clinical signs rather than treating the cause itself, which remains elusive. There are a plethora of treatments recommended and utilized for management of headshaking; however, reported rates of success of such treatments are rare and often contain limited numbers of horses. This study describes the owner-reported outcome of 11 treatments used in the management of idiopathic headshaking in 130 horses.

The demographics of the survey population and prevalence of clinical signs were similar to that described in other headshaking studies. Some variation in observed clinical signs existed between horses diagnosed by owners and veterinarians, with the latter group representing a more severely affected cohort, as previously reported. Owners have proven reliable at using the clinical signs of vertical headshaking, rubbing the nose on the thoracic limb, and snorting for the diagnosis of idiopathic headshaking. As vertical headshaking was observed in over 90% of horses in this study, it is likely that owner-diagnosed horses were true idiopathic headshakers; however, other causes of headshaking cannot be excluded. Likewise, details of the veterinary diagnostic procedure of the veterinarian-diagnosed horses are also unknown. In field situations, veterinarian diagnosis of headshaking, like owner assessment, is usually based on observation and history. A single veterinary exam has limitations, and there is no diagnostic test for headshaking. Idiopathic headshaking is a constellation of signs, which persist in the absence of a rider, independent of tack, and apparent lack of underlying pathology (i.e., sinus mass, iris cyst, etc.). The authors, therefore, feel that veterinary diagnosis and owner assessment of clinical signs, which are the basis of any headshaking diagnosis, are both equally valid.

Nose nets were the most frequently used treatment for headshaking having been used by almost 90% of respondents. Whereas some degree of improvement was seen in over 50% (58/110) of horses, complete resolution of headshaking was rare and observed in only 5%. These improvements are lower than that previously reported where use of nose nets resulted in some decrease in clinical signs in 75% of 36 headshakers in the UK but greater than the 33% improvement reported in the U.S. Geographical location was not analyzed in this study due to the heterogeneous nature of the data. In this study, nose nets resulted in one of the highest variation in observed clinical signs existed between horses diagnosed by owners and veterinarians, with the latter group representing a more severely affected cohort, as previously reported. Owners have proven reliable at using the clinical signs of vertical headshaking, rubbing the nose on the thoracic limb, and snorting for the diagnosis of idiopathic headshaking. As vertical headshaking was observed in over 90% of horses in this study, it is likely that owner-diagnosed horses were true idiopathic headshakers; however, other causes of headshaking cannot be excluded. Likewise, details of the veterinary diagnostic procedure of the veterinarian-diagnosed horses are also unknown. In field situations, veterinarian diagnosis of headshaking, like owner assessment, is usually based on observation and history. A single veterinary exam has limitations, and there is no diagnostic test for headshaking. Idiopathic headshaking is a constellation of signs, which persist in the absence of a rider, independent of tack, and apparent lack of underlying pathology (i.e., sinus mass, iris cyst, etc.). The authors, therefore, feel that veterinary diagnosis and owner assessment of clinical signs, which are the basis of any headshaking diagnosis, are both equally valid.

Nose nets were the most frequently used treatment for headshaking having been used by almost 90% of respondents. Whereas some degree of improvement was seen in over 50% (58/110) of horses, complete resolution of headshaking was rare and observed in only 5%. These improvements are lower than that previously reported where use of nose nets resulted in some decrease in clinical signs in 75% of 36 headshakers in the UK but greater than the 33% improvement reported in the U.S. Geographical location was not analyzed in this study due to the heterogeneous nature of the data. In this study, nose nets resulted in one of the highest variation in observed clinical signs existed between horses diagnosed by owners and veterinarians, with the latter group representing a more severely affected cohort, as previously reported. Owners have proven reliable at using the clinical signs of vertical headshaking, rubbing the nose on the thoracic limb, and snorting for the diagnosis of idiopathic headshaking. As vertical headshaking was observed in over 90% of horses in this study, it is likely that owner-diagnosed horses were true idiopathic headshakers; however, other causes of headshaking cannot be excluded. Likewise, details of the veterinary diagnostic procedure of the veterinarian-diagnosed horses are also unknown. In field situations, veterinarian diagnosis of headshaking, like owner assessment, is usually based on observation and history. A single veterinary exam has limitations, and there is no diagnostic test for headshaking. Idiopathic headshaking is a constellation of signs, which persist in the absence of a rider, independent of tack, and apparent lack of underlying pathology (i.e., sinus mass, iris cyst, etc.). The authors, therefore, feel that veterinary diagnosis and owner assessment of clinical signs, which are the basis of any headshaking diagnosis, are both equally valid.

Nose nets were the most frequently used treatment for headshaking having been used by almost 90% of respondents. Whereas some degree of improvement was seen in over 50% (58/110) of horses, complete resolution of headshaking was rare and observed in only 5%. These improvements are lower than that previously reported where use of nose nets resulted in some decrease in clinical signs in 75% of 36 headshakers in the UK but greater than the 33% improvement reported in the U.S. Geographical location was not analyzed in this study due to the heterogeneous nature of the data. In this study, nose nets resulted in one of the highest variation in observed clinical signs existed between horses diagnosed by owners and veterinarians, with the latter group representing a more severely affected cohort, as previously reported. Owners have proven reliable at using the clinical signs of vertical headshaking, rubbing the nose on the thoracic limb, and snorting for the diagnosis of idiopathic headshaking. As vertical headshaking was observed in over 90% of horses in this study, it is likely that owner-diagnosed horses were true idiopathic headshakers; however, other causes of headshaking cannot be excluded. Likewise, details of the veterinary diagnostic procedure of the veterinarian-diagnosed horses are also unknown. In field situations, veterinarian diagnosis of headshaking, like owner assessment, is usually based on observation and history. A single veterinary exam has limitations, and there is no diagnostic test for headshaking. Idiopathic headshaking is a constellation of signs, which persist in the absence of a rider, independent of tack, and apparent lack of underlying pathology (i.e., sinus mass, iris cyst, etc.). The authors, therefore, feel that veterinary diagnosis and owner assessment of clinical signs, which are the basis of any headshaking diagnosis, are both equally valid.

Nose nets were the most frequently used treatment for headshaking having been used by almost 90% of respondents. Whereas some degree of improvement was seen in over 50% (58/110) of horses, complete resolution of headshaking was rare and observed in only 5%. These improvements are lower than that previously reported where use of nose nets resulted in some decrease in clinical signs in 75% of 36 headshakers in the UK but greater than the 33% improvement reported in the U.S. Geographical location was not analyzed in this study due to the heterogeneous nature of the data. In this study, nose nets resulted in one of the highest variation in observed clinical signs existed between horses diagnosed by owners and veterinarians, with the latter group representing a more severely affected cohort, as previously reported. Owners have proven reliable at using the clinical signs of vertical headshaking, rubbing the nose on the thoracic limb, and snorting for the diagnosis of idiopathic headshaking. As vertical headshaking was observed in over 90% of horses in this study, it is likely that owner-diagnosed horses were true idiopathic headshakers; however, other causes of headshaking cannot be excluded. Likewise, details of the veterinary diagnostic procedure of the veterinarian-diagnosed horses are also unknown. In field situations, veterinarian diagnosis of headshaking, like owner assessment, is usually based on observation and history. A single veterinary exam has limitations, and there is no diagnostic test for headshaking. Idiopathic headshaking is a constellation of signs, which persist in the absence of a rider, independent of tack, and apparent lack of underlying pathology (i.e., sinus mass, iris cyst, etc.). The authors, therefore, feel that veterinary diagnosis and owner assessment of clinical signs, which are the basis of any headshaking diagnosis, are both equally valid.

Nose nets were the most frequently used treatment for headshaking having been used by almost 90% of respondents. Whereas some degree of improvement was seen in over 50% (58/110) of horses, complete resolution of headshaking was rare and observed in only 5%. These improvements are lower than that previously reported where use of nose nets resulted in some decrease in clinical signs in 75% of 36 headshakers in the UK but greater than the 33% improvement reported in the U.S. Geographical location was not analyzed in this study due to the heterogeneous nature of the data. In this study, nose nets resulted in one of the highest variation in observed clinical signs existed between horses diagnosed by owners and veterinarians, with the latter group representing a more severely affected cohort, as previously reported. Owners have proven reliable at using the clinical signs of vertical headshaking, rubbing the nose on the thoracic limb, and snorting for the diagnosis of idiopathic headshaking. As vertical headshaking was observed in over 90% of horses in this study, it is likely that owner-diagnosed horses were true idiopathic headshakers; however, other causes of headshaking cannot be excluded. Likewise, details of the veterinary diagnostic procedure of the veterinarian-diagnosed horses are also unknown. In field situations, veterinarian diagnosis of headshaking, like owner assessment, is usually based on observation and history. A single veterinary exam has limitations, and there is no diagnostic test for headshaking. Idiopathic headshaking is a constellation of signs, which persist in the absence of a rider, independent of tack, and apparent lack of underlying pathology (i.e., sinus mass, iris cyst, etc.). The authors, therefore, feel that veterinary diagnosis and owner assessment of clinical signs, which are the basis of any headshaking diagnosis, are both equally valid.
that previously reported\(^7,9\) and equivalent to use of a nose net in this study, although 20% fewer respondents had tried them. Again, adverse effects not previously reported such as “spookiness” and stumbling due to presumed reduced vision were frequent and observed in over ¼ of horses using face masks. Whereas observed less frequently than with use of a nose net, these reported adverse effects were of a more disruptive nature.

A surprisingly large percentage of horses (21%; 18/84) were reported to derive some benefit from the use of fly control, in contrast to only 2% previously described.\(^7\) The current study population may have comprised more mildly affected horses than the aforementioned study,\(^7\) despite ⅜ of owners describing their horse’s headshaking as “severe” or “very severe.” It is likely that a positive outcome rate of approximately 20% is heavily influenced by response bias, as no treatment achieved lower than 18% positive outcome. Such bias may have arisen from multiple sources including the difficulty in objective assessment of headshaking and any treatment response, a proxy-placebo effect, the phenomena of spontaneous and seasonal remission, fluctuation of clinical signs, regression to the mean, any contemporaneous treatments, and recall bias. Whereas such bias limits interpretation of all observed responses to treatments, the variability in reported outcomes gives confidence that a genuine treatment effect, above and beyond any bias, was noted for some treatments.

The positive response of over 50% (17/31) of headshakers to corticosteroid therapy was also unexpected given the documented aberrant trigeminal nerve activity\(^3\) and the failure of a recent blinded, clinical trial of pulsed high dose dexamethasone therapy to have any effect on idiopathic headshaking.\(^15\) Whereas not a first-line treatment for neuropathic pain, corticosteroids have been shown to reduce spontaneous discharge in injured nerves and might be useful as adjunct therapy.\(^16\) Although nerve injury is not suspected in idiopathic headshaking, a reduced threshold for activation of the trigeminal nerve has been documented\(^6\) and corticosteroids might act to decrease such aberrant activity. It is also possible that, in the responding subset of headshaking horses, there was a degree of low grade inflammation or allergy contributing to clinical signs observed by owners. As prolonged, ongoing management of headshaking is usually required, corticosteroids are unlikely to be useful as a therapeutic option due to the risk of serious adverse effects such as laminitis.

One-third of owners reported general antihistamine treatment improved their horse’s headshaking (12/36). Such improvement may suggest an allergenic component to headshaking in these horses; however, the reported effect may be largely due to bias as previously discussed. A positive response to cyproheptadine treatment was observed in approximately 50% of horses (14/29). Cyproheptadine is a first generation antihistamine with additional anticholinergic, antiserotonergic, calcium channel blocking, and local anesthetic activity,\(^17\) which is used to treat human vascular headaches. Previous reports describing the response of headshaking to cyproheptadine treatment has varied widely, from no improvement\(^4,18\) to 70%.\(^7\) Such variation in efficacy is difficult to explain. The percentage of horses with a photic component to their headshaking is variable across studies (none\(^4\); 39%, current study; 60%\(^7\)), which may be relevant in responsiveness to cyproheptadine. Inappropriate dosage may have affected treatment outcome with cyproheptadine in the current study as, whereas only 2 owners reported using an inadequate dosage, many owners reported they did not know or omitted administration details. Adverse effects such as lethargy and drowsiness were noted in almost 50% of horses treated with cyproheptadine, which is likely to limit its use in ridden horses.

Carbamazepine, an anticonvulsant, which stabilizes voltage-gated sodium channels, had one of the lowest positive outcome rates of all treatments (25%; 2/8) and, therefore, does not appear to be an efficacious treatment for headshaking. Additionally, negative side effects were reported in ⅘ of treated horses. Although carbamazepine is the treatment of choice for human trigeminal neuralgia,\(^19\) the pharmacokinetics of this drug in horses is unknown, which may explain its lack of effect. Additionally, recent trigeminal electrophysiological data suggests the etiopathogenesis of equine headshaking and human trigeminal neuralgia may differ.\(^5\) NSAIDs were also poorly efficacious (22%; 4/22) as would be expected in the treatment of a neuropathic condition. The noted response in 4 horses may be due to the aforementioned biases.

Melatonin reduced headshaking in almost half (47%; 8/17) of horses in this study. The hypothesis for melatonin treatment of headshaking is that a late afternoon (5 PM) dose of melatonin artificially manipulates photoperiod such that, biologically, the horse remains in winter. Approximately 40% of treated horses did not shed their coat due to this phenomenon. Therapy is proposed to be most successful when melatonin is started before the onset of spring for seasonal headshakers and, therefore, initiation of therapy when the horse was already headshaking may have reduced efficacy. A positive outcome in the two horses given melatonin inappropriately and the fact that suppression of the vernal increase in gonadotropins did not reduce headshaking\(^20\) may question the proposed rationale. However, melatonin is also able to modulate pain, including neuropathic pain, via various antinociceptive effects including activation of opioid receptors, inhibition of pro-inflammatory cytokine production, modulation of GABA\(_A\) receptor function and acting as a free radical scavenger.\(^21\) Melatonin receptors have been identified in the trigeminal ganglion and trigeminal nucleus of mammals\(^22\) and interestingly,
Melatonin has been shown to attenuate an inducible model of trigeminovascular nociception in rats. Melatonin might, therefore, act to reduce headshaking by mechanisms other than photoperiod manipulation. Only 13% of owners had used melatonin treatment, and all except one of these were based in North America, where the drug is cheaply available as an “over the counter” product. Whereas melatonin can be purchased with a veterinary prescription in other countries such as the UK, this does not appear to be occurring, most likely due to veterinarians being currently unaware of the protocol. Melatonin, as an endogenous hormone, also has the advantage of having no restrictions for use in competition horses. Given that almost half of headshakers improved with melatonin therapy, with only winter coat retention as an adverse effect, this treatment warrants further attention.

Magnesium supplementation had been used by almost 50% of owners, with just under half of these (25/58) reporting an improvement in their horse’s headshaking. Magnesium increases the activation threshold of nerves such that a greater stimulus is required for depolarization. Magnesium, therefore, appears a rational therapy given the reduced activation threshold of the trigeminal nerve in headshaking horses. Formulations of magnesium vary in their oral bioavailability with that of magnesium oxide (the most frequently administered in this study; however, the number of horses using these therapies was low and, therefore, these results should be interpreted with caution. Of these pharmaceutical drugs, only melatonin and melatonin therapy appeared to be occurring, most likely due to veterinarians being currently unaware of the protocol. Melatonin, as an endogenous hormone, also has the advantage of having no restrictions for use in competition horses. Given that almost half of headshakers improved with melatonin therapy, with only winter coat retention as an adverse effect, this treatment warrants further attention.

This study can be used to guide owners in the management of idiopathic headshaking. Owner-reported responses to treatment in 130 headshaking horses suggest that nose nets, face masks, and magnesium supplementation are the most efficacious nonmedical therapeutic options. Adverse effects such as facial irritation were reported in 1/5 of horses with nose nets. Cyproheptadine, corticosteroids, and melatonin were the most successful pharmaceutical treatments in this study; however, the number of horses using these therapies was low and, therefore, these results should be interpreted with caution. Of these pharmaceutical drugs, only melatonin is without significant adverse effects or allowed in competition horses. Veterinary diagnosis of idiopathic headshaking is advised before commencing any treatments. Further investigation of the pathophysiological mechanism underlying the aberrant trigeminal nerve activity in headshaking horses is warranted in order to develop more successful therapeutic options.

Acknowledgments

Conflict of Interest

This study was funded by anonymous private donation to the Equine and Comparative Neurology Research Group.

References and Footnotes


*Survey Monkey, Palo Alto, CA 94301-2580.
Facebook, Menlo Park, CA 94025.*
Review of the Discovery of the Basis for a Seasonal Pasture Myopathy/Atypical Myopathy

Stephanie Valberg, DVM, PhD, DACVIM, DACVSMR

The amino acid hypoglycin A is present in the seeds of box elder and sycamore maple trees. When pastured horses ingest seeds in the fall and early spring they develop a highly fatal form of rhabdomyolysis. A definitive diagnosis is made by measurement of serum acylcarnitines, which accumulate because the toxin blocks muscle lipid metabolism. Author's address: Department of VPM, University of Minnesota, 1965 Gortner Avenue, St. Paul, MN 55108; e-mail: valbe001@umn.edu. © 2014 AAEP.

1. Introduction
Seasonal pasture myopathy,1,2 atypical myopathy,3,4 and atypical myoglobinuria5 are terms used to describe an often-fatal form of rhabdomyolysis that develops in unexercised horses kept on pastures. Although initial clinical signs resemble colic, the apparent discomfort, trembling, tachycardia, short stiff stride, periods of recumbency, tachypnea, and myoglobinuria actually arise from severe degeneration of postural and respiratory muscles.5–7 The first reports date back to 1939 in Wales8 and 1959 in Southern Ontario.9 Although described for 75 years, the cause of this disorder has remained a mystery until very recently. The purpose of this review is to highlight the sentinel findings that led to the discovery of the toxin associated with cases of seasonal pasture myopathy in European and North American horses.

2. Epidemiology
Vital epidemiological information on a large number of cases of pasture myopathy arose from the Atypical Myopathy Alert Group (http://www.myopathieaty-pique.fr/en/amag-2/). Many hundreds of cases were documented in Denmark, France, Germany, Ireland, United Kingdom, Latvia, Luxembourg, Spain, Switzerland, The Netherlands, Austria, Czech Republic, Italy, Norway, and Sweden.7,10–12 Fewer carefully researched cases were documented in North America.1,2,13 It became apparent that the number of outbreaks of pasture myopathy varied from year to year, were preceded by stormy weather, and occurred in pastures with trees in or around them that also contained dead leaves, dead wood and wet areas. The clustering of cases to specific pastures where horses were housed for at least 12 h a day, the seasonal occurrence in fall or the subsequent spring, the cessation of cases from December to January, and the lack of gender or breed predilection suggested a toxin was present in pastures on a seasonal basis. The wide variety of plants that could be implicated in causing pasture myopathy made the search for potential toxins confounding. Potential etiologic agents initially under investigation included white snakeroot,1 clostridium toxins in soil,14,15 toxins in dead leaves,10,12 and the tar spot fungus growing on the leaves of Acer pseudoplatanus,16 the European sycamore maple. Studies from 1988 in the United Kingdom5 and
2010 in the Netherlands had identified the sycamore maple as a consistent presence on affected pastures. Information on the mechanism by which the toxin produced rhabdomyolysis was an important missing piece of the puzzle.

3. Pathophysiology
Serum biochemistry profiles of horses with pasture myopathy provided little direction for toxin investigation. Profiles revealed marked elevations in serum creatine kinase (CK) and aspartate transaminase and a moderate elevation in gamma-glutamyl transferase activities. Electrolyte derangements (high potassium and phosphorus, low sodium, chloride, and calcium), hyperglycemia, and lactic acidosis were often present. Similarly, hematoxylin and eosin stains of formalin fixed muscle merely confirmed severe myodegeneration of postural and respiratory muscles and inconsistent degeneration of cardiac muscle. Advances were made, however, when special stains were applied to muscle tissues and a lipid storage disorder was identified in postural and respiratory muscles of horses with pasture myopathy in both North America and Europe.

A critical piece of the puzzle was contributed by Westerman et al. in 2008 that both narrowed the list of potential toxins causing pasture myopathy and led to a specific means to diagnose the condition. An acquired defect in the enzyme multiple acyl-coA dehydrogenase (MAD) that represents the first step of \( \beta \)-oxidation of short and medium chain fatty acids as well as branched chain amino acids was found in muscle cells from Dutch horses with pasture myopathy. A diagnosis of MAD deficiency could readily be established by measuring the backlog of byproducts of lipid (acylcarnitines) and amino acid metabolism (glycine conjugates) in the bloodstream or urine. The specific diagnostic indicators of MAD were high short (C2-C5) and medium chain (C6-C10) acylcarnitines, variably high long chain (C14-C18) acylcarnitines, and marked elevations in urine ethylmalonic and methylsuccinic acids and glycine conjugates (isovaleryl-, butyryl-, and hexanoylglycine). Serum and urine samples from horses in North America with pasture myopathy were later found to also have a deficiency of MAD. Thus, it seemed that the underlying mechanism for pasture myopathy was the same in Europe and North America.

4. The Toxin
Combining epidemiologic and pathophysiologic information, the responsible toxin would have to be seasonally associated with trees and capable of blocking MAD in muscle cells. In 2012, we determined that seeds from one particular tree in the midwestern U.S., Acer negundo (box elder, Manitoba maple) (Fig. 1A), were present in the fall on all inspected pastures where horses had succumbed to pasture myopathy. The seeds of this tree (Fig. 1B) remain on the trees in the fall and are distributed onto the ground by high winds prior to an outbreak. The topographical distribution of Acer negundo matched previous cases of pasture myopathy diagnosed in North America. A key connector was the fact that another tree within the same Sapinidae family, the Jamaican Ackee tree, produced a fruit that when unripe contained a nonproteogenic amino acid hypoglycin A, capable of causing an acquired deficiency of MAD when ingested by humans. Subsequent studies found hypoglycin A in seeds from box elder trees growing in pastures where outbreaks of pasture myopathy had occurred. In addition, the conjugated toxic metabolite of hypoglycin A, methyleneacyclopropyl acetic acid (MCPA)-carnitine, was found in the bloodstream and urine of horses with pasture myopathy in the midwestern U.S. Thus, hypoglycin A appeared to cause pasture myopathy in North America.

Fig. 1. A, Branches of a box elder tree (Acer negundo) laden with seeds in the late fall. B, One samara (husk) from the box elder tree with the seed removed (above and to the left) relative to the size of a razor blade (below).
Acer negundo was an unlikely source of toxin in Europe as this tree is not native to Europe and it was introduced largely as an ornamental varietal. A closely related tree, Acer pseudoplatanus, the sycamore maple (Fig. 2A), had previously been identified in European pastures where outbreaks occurred. Hypoglycin A was analyzed and identified in seeds from Swiss sycamore trees (Fig. 2B) that were growing in pastures where horses had died from pasture myopathy and blood samples from affected European horses were also found to contain serum MCPA-carnitine.21 Thus, hypoglycin A present in seeds obtained in the fall from box elder or sycamore maples is the likely etiology for pasture myopathy in North America and Europe. Storms increase the number of seeds on pastures;2,10,11 snow cover in the winter prevents access to seeds on the ground and ingestion of germinating seeds in the early spring and can lead to emergence of new cases. Seed burdens show annual variation, potentially explaining variations in outbreaks from year to year.22 Toxin levels in seeds could vary from year to year as well, but this has not been studied. Disruption of lipid metabolism in muscle cells leads to degeneration of those muscle cells most active at rest, postural and respiratory muscles.

No studies have been performed in which hypoglycin A or Acer species seeds have been fed to horses to definitively prove the toxicity of Acer species seeds in horses. Thus, the number of seeds a horse would need to eat to become ill is not known. Further, quantitative assays of hypoglycin A have not been performed on seeds from other maple trees to determine their toxicity, nor have changes in hypoglycin A concentrations been reported over the course of a year.

5. Diagnosis
A presumptive diagnosis of hypoglycin A-induced pasture myopathy is based on the following:

1. Clinical signs of sudden rapidly progressive muscular weakness and stiffness, myoglobinuria, tachycardia, tachypnea, dyspnea, and recumbency, with death occurring in the majority of cases within 72 hours of the onset of signs. About 50% of horses will have cardiac damage. Most horses retain a normal appetite; occasionally horses have signs of choke, cardiac arrhythmia, colon impactions, and head swelling due to persistently low head posture.

2. Marked elevations in serum CK and aspartate transaminase activities, hyperglycemia, electrolyte derangements, and acidosis.

3. A history of grazing pastures containing or surrounded by maple trees with seeds in the fall or early spring.

A definitive diagnosis of hypoglycin A-induced myopathy is based on characteristic changes in serum acylcarnitines and urine organic acids. These tests are most readily available and are routinely performed by human metabolic disease laboratories such as the Baylor Institute of Metabolic Disease (http://www.baylorhealth.edu/Research/Institutes-Centers/IMD/Pages/Tests%20and%20Forms%2005-11.pdf). Normal equine values have been published for reference ranges.13 Hypoglycin A and MCPA-carnitine assays are not commercially available.

6. Treatment
Early recognition of the disease is critical as the mortality rate likely improves with early treatment and prompt removal of horses from the source of toxin. Since this condition can present as a succession of cases, removal of all grazing animals from the pasture immediately is important along with checking serum CK activities. Horses should be rested in a warm dry environment and fed ad libitum hay and some grain to avoid a negative energy balance. Renal, respiratory, and cardiac function should be monitored by means of serum biochemistry profiles, blood gases, and cardiac troponin. Judicious use of nonsteroidal anti-inflammatory drugs is recommended, based on the status of dehydration.
and renal function. Intravenous fluids need to be tailored to each horse’s electrolyte derangements and severity of lactic acidosis at a rate that avoids fluid overload, especially if the cardiovascular system is compromised. Nasal oxygen may benefit horses with tachypnea. Intravenous antioxidants such as dimethyl sulfoxide, vitamin C, vitamin B complex (including riboflavin), and oral water dispersible natural vitamin E may be beneficial for the outcome.1,6 The mortality rate is about 75%.10 Horses that have recovered usually regain muscle mass over time and return to their former level of activity.

7. Prevention
Minimizing a horse’s risk of exposure to box elder or European sycamore seeds in the fall is the best preventive strategy. Nevertheless, it is important to recognize that there are many older horses that have been housed on pastures with large numbers of Acer trees for years that have never become ill. Whether these horses avoid eating the seeds or have some form of immunity is unclear. Horses particularly at risk for pasture myopathy are those that are young or new to overgrazed pasture with Acer trees, those turned out more than 12 h/day, and those receiving little supplemental feed.2,10 Ideally, trees with heavy seed burdens should be removed from pastures. If this is not possible, turn-out of at-risk horses should be avoided in the fall when seed burdens are high or in the early spring.

8. Name Change for Atypical Myopathy and Seasonal Pasture Myopathy
There are other potential causes for pasture-associated myopathies in horses such as trematone toxin in white snakeroot and rayless goldenrod. With advances in defining the etiology of the most common pasture myopathy, the terms Seasonal Pasture Myopathy and Atypical Myopathy are now outdated and nonspecific. A change to the name Hypoglycin A Myopathy (H-AM) is proposed for those cases in which rhabdomyolysis develops in horses that are housed in proximity to seed-bearing Acer seeds or Acer pseudoplatanus trees, especially when elevated serum acylcarnitine or urine glycine conjugates concentrations are confirmed to be present.

Acknowledgments
This research represents a collaborative effort amongst researchers at the University of Bern, Switzerland, the University of Liege, Belgium, Institute of Metabolic Disease, Baylor Research Institute, Texas, Iowa State University, and the Department of Horticultural Sciences, University of Minnesota.

Conflict of Interest
This research was funded by the University of Minnesota Rapid Agricultural Response Fund.

References
Effect of a Dietary Supplement on Gastric Ulcer Severity

Nicola Kerbyson, BVMS, Cert AVP (EM), MRCVS*; Derek Knottenbelt, OBE, BVM&S, DECEIM, MRCVS; and Tim Parkin, BSc, BVSc, PhD, DECVP, FHEA, MRCVS

There is no statistically significant difference in the effect of omeprazole or a dietary supplement in the treatment of squamous gastric ulceration in horses in active race training, with neither treatment resulting in improved ulcer scores following 90 days of treatment. Authors’ address: University of Glasgow, Scotland, UK G61 1QH; e-mail: n.kerbyson.1@research.gla.ac.uk. *Corresponding and presenting author. © 2014 AAEP.

1. Introduction
The objective of this study was to assess the effect of a dietary supplement containing polar lipids (oat oil), soluble fiber (β-glucan), and amino acids (L-Glutamine and L-Threonine) on the development and treatment of squamous gastric ulceration in racing Thoroughbreds.

2. Materials and Methods
Sixty-seven Thoroughbreds in race training or pre-race training were recruited to the study on the basis of having grade 1 gastric ulceration (The Equine Gastric Ulcer Council, 1999) on day 0 of the trial. Of this group, 39 horses completed the trial, which involved repeat gastroscopy on day 30, 60, and 90. Each horse was randomly assigned to one of two treatment groups; omeprazoleb or the dietary supplementa; treatments were administered according to label dosage for the duration of the trial.

3. Results and Discussion
Chi-squared tests were used to assess the difference between treatment groups in terms of the proportion of horses improving by 2 or more grades: sixteen horses in each group improved by less than 2 grades or worsened (P = 0.53). McNemar’s tests were applied to assess the effect of each treatment on gastric ulcer score. There was no significant improvement on gastric ulcer score with either omeprazole (P = 0.38) or the dietary supplement (P = 0.50) when a clinically significant gastric ulcer score of ≥2 was used.

Acknowledgments
Conflict of Interest
This study was funded by Freedom Health LLC.

Footnotes
aSucceed™, Freedom Health LLC, Aurora, OH 44202.
bGastroGard®, Merial Animal Health Ltd., Harlow, Essex, UK.
Effect of a Supplement on Non-Glandular Gastric Ulcer Scores and Gastric Juice pH

Frank M. Andrews, DVM, MS, DACVIM-LA; Pilar Camacho, DVM; Giavanna Gaymon, DVM; Patrick Loftin, DVM; Frank Garza, Jr., MS; Michael L. Keowen, BS; and Michael T. Kearney, PhD

A supplement (SGU) a added to feed prevents the worsening of nonglandular gastric ulcers in stall-confined horses 2 weeks after discontinuation of omeprazole treatment and lessens the increase in gastric ulcer scores after intermittent feeding, without altering the gastric juice pH. Authors' addresses: Equine Health Studies Program, Louisiana State University, School of Veterinary Medicine, Baton Rouge, LA 70803 (Andrews, Camacho, Loftin, Garza, Keowen, Kearney); and Tuskegee University School of Veterinary Medicine, Tuskegee, AL 36088 (Gaymon); e-mail: fandrews@lsu.edu. © 2014 AAEP.

1. Introduction
SGU containing botanicals (herbs), coating agents, and natural antacids for horses with gastric ulcers has limited data available on its effectiveness. The purpose of this study was to evaluate the effect of SGU on nonglandular (NG) gastric ulcers scores and gastric juice pH after omeprazole treatment in stall-confined horses.

2. Materials and Methods
This study was performed in racing-age stall-confined Thoroughbreds (n = 8) as a 42-day 2-period crossover design in which all horses received no treatment (untreated controls) or treatment (SGU pellets, 40 g, twice daily). In addition, from days 1 to 14 all horses were treated with omeprazole paste b, and then treatment was discontinued. From days 28 to 35, all horses underwent intermittent feed-deprivation. During feed-deprivation, all treated horses were fed SGU. From days 35 to 42, horses returned to their normal diet. Gastroscopy was performed on day −1, 14, 28, 35, and 42. Gastric juice pH was measured and NG gastric ulcer number (NGN) and severity (NGS) scores were assigned by a masked clinician (FMA).

3. Results
After 14 days, NGN and NGS scores significantly decreased (P < 0.05) in both groups compared to those on day −1. On days 28 and 35, NGN scores remained significantly lower in the SGU-treated horses when compared to the untreated controls. By Day 42, NGN and NGS scores were not significantly different in either group. Gastric juice pH was low and variable, except on day 14, when gastric juice pH was significantly higher in both groups due to omeprazole treatment.

4. Conclusions
SGU supplement fed to horses prevented gastric ulcers from increasing in horses after omeprazole treatment.
Medicine Potpourri: Neurology, Foals, Muscles, and the Gut

Treatment, without increasing gastric juice pH. Supplementation with SGU aids in the protection of the NG stomach from the rebound acid effects after omeprazole treatment is discontinued and in stall-confined horses undergoing intermittent feeding.

Acknowledgments

Conflict of Interest

This study was funded by Smart Pak Equine, LLC., Equine Health Studies Program Equine Funds and the LSU Foundation.

Footnotes

aSmartGut® Ultra, SmartPak Equine, LLC., Plymouth, MA 02360.
bGastroGard® paste, Merial Limited, Duluth, GA 30096.